CASE STUDY : FADING ELK SYNDROME AT INVERMAY

C.G. Mackintosh, MAF Technology, Invermay, Private Bag, Mosgiel
M.B. Orr, MAF Quality Assurance, Invermay AHL, Private Bag, Mosgiel

Introduction

The "fading elk syndrome" is characterised by chronic weight loss, illthrift and general lack of response to broad treatment. It has been a common problem in many elk (wapiti) herds throughout this country since the animals were imported less than 10 years ago. Initially there was under-reporting of the condition due to embarrassment of all the and concern that it gave elk a "bad name". However, more recently farmers and vets have been more willing to acknowledge that elk have specific health related problems. Over the last 9 years we have had a number of cases of "fading elk" at Invermay and an associated Landcorp property, Orokonui, and these were presented at the 1988 Deer Branch Course No. 5 by A.J. Pearse in his paper on the special management needs of wapiti and hybrids. The following case study is a collection of reports of all the Invermay owned elk or elk x red hybrids which showed clinical signs of weight loss and death during the latter half of 1989 on three farms, Invermay, Waiora and Orokonui. They provide a basis for discussion of possible causes, preventative measures and treatments.

Case histories

16 June:	Waiora. P29, pregnant 10 year old imported Canadian elk hind found dead after appearing "off colour" but not scouring and losing weight for a few days. Feeding on grass and lucerne hay.
P.M.:	Hind in good condition. Torsion of abomasum, hugely distended with dry ingesta, peritonitis and haemorrhagic gastroenteritis.
Diagnosis:	Abomasal torsion. Aetiology not known.
24 July:	Waiora. P803, 7 month old male elk weaner found dead, after being "off-colour" for one week. Small for its age. Given long-acting penicillin prior to death.
P.M.:	No body fat, rumen contained one-third normal volume of dry ingesta, fluid in intestinal tract but faeces pelleted. Milky fluid, 200-300 mls in abdominal cavity. Beneath tongue embedded in muscle tissue was a small thick-walled abscess (1 cm diameter) containing creamy pus. No other gross abnormalities. The abscess was negative for AFO's but contained large numbers of Gram -ve organisms, and a small number of Gram +ve organisms.
Diagnosis:	Sublingual abscess inhibiting eating and swallowing together with possible toxic effects.

22 August: Waiora. BY415, 4 year old F1 elk x red hybrid stag found dead after appearing off colour for 3 or 4 days. Had been taken out of large group and put in small grassy paddock with another stag for company. In light condition.

P.M.: Severe oedema of mesentery and abomasum. Rumen and abomasal lymph nodes hard and knobbly. Some mottling of the lungs. Numerous lungworm in bronchi. Abomasal worm count revealed 13,600 Ostertagia-type and 2,400 Trichostrongyle-type worms present. Histopathology showed severe oedema of gut serosa and mesentary. Numerous lungworms in bronchi.

P.M.: Severe abomasal parasitism.

23 August: Invermay. P14, 10 year old imported Canadian elk bull died after prolonged illness. Initially had diarrhoea in May after the rut, treated with Ivomec, vitamins and parenteral long acting tetracyclines. Scour ceased but animal had poor appetite and was losing condition. Kept in yards. Offered ad libitum lucerne hay. Ate small quantity crushed barley daily. Sparse faeces. Continued to lose weight. Euthanased.

P.M.: Grossly distended abomasum, 45 cm diameter, with dry compacted contents, small amount of liquid contents in rumen, omasum, pyloric end of abomasum and small intestine. Sparse rectal faeces.

Diagnosis: Abomasal impaction. No obvious displacement.

September: Waiora. Elk weaners in mixed group at pasture not doing as well as expected. Faecal samples taken from 2 elk, 2 F1 and 4 red weaners at random. Had low lungworm larvae (up to 2.4 L_1/gm) and negative faecal egg counts. All weaners were drenched with oral Ivomec and given 4 g capsules of copper needles.

2 October: Waiora. BY724, 2 year old F1 hind found rubbing head on post, very swollen eyelids, obvious photosensitivity, yellow mucous membranes, poor condition, loose faeces, elevated temperature, distressed and was euthanased after blood samples taken.

P.M.: Very yellow carcass, rounded liver margins. Histopathology showed recent severe hepatotoxic damage, also quite a few nematode larvae in abomasal mucosa and early acute bronchiolitis. Serum biochemistry showed raised GGT, GLDH, bilirubin and low serum albumin and total protein (see Table 1).

Diagnosis: Severe acute liver disease and obstructive jaundice. Also, abomasal parasitism.

6 October: Waiora. Herd mates of BY724 (jaundice case) examined. A third of the group of 20 animals were in poor condition. Five worst animals were sampled (BY727, 728, 730, 733 and BY 647). Four of the five had soft or very soft faeces, one had slightly elevated egg count (500 e/g), the rest negative. All were negative for titres to Leptospira pomona and L. copenhageni. All had low serum albumin levels. Three had evidence of liver damage with varying elevations in serum GGT, GLDH and/or bilirubin levels (see Table 1).

The majority of animals gradually improved except for one BY733 which "faded" and was euthanased in December. Necropsy showed signs of hepatotoxic damage, mild focal hyperplasia and submucosal oedema of the abomasum and very little colloid in the thyroid gland (hypoplastic).

16 October: Waiora. P806, 10 month old female elk weaner in very thin condition was found hiding in paddock, very weak. Sampled and died shortly after.

P.M.: Abomasal worm count and faecal egg count, negative. Liver copper 930 μmol/kg (adequate). Marked serosal and submucosal oedema of small intestine. Moderate chronic colitis and serosal oedema of large intestine. Very mild changes in hepatocytes near portal triad and some bile retention. Biochemistry showed highly elevated SGOT, GGT and GLDH indicating severe liver disease. Very low serum albumin (see Table 1).

Diagnosis: Liver disease.

17 October: Waiora. P802, 10 month old male elk weaner (herd mate of P806) in poor condition brought into yards. Treated with Ivomec, fluids, antibiotics and vitamins. Kept indoors for 2 months convalescence on hay and concentrates. Biochemistry revealed severe liver damage and inflammation (see Table 1). These parameters improved gradually over the next month.

Diagnosis: Liver disease.

19 October: Waiora. The group of rising yearlings from which P806 and P802 came were yarded and blood samples were taken from 3 pure, 2 seven-eighths, 3 three-quarters, 3 half and 3 quarter elk animals, along with a Pere David's x red hybrid. These were compared with 5 rising 2 year old pure red hinds in a neighbouring paddock. The majority were in moderately good condition but the pure elk and some of the others were in light condition although none appeared to be scouring. All were grazing on mixed, predominantly ryegrass/clover pasture. The blood biochemistry results showed mildly elevated GLDH and GGT values indicative of mild liver

damage in a number of animals especially the elk and elk hybrids (see Table 2).

10 November: Waiora. Repeat blood samples taken from the above animals showed that most values had returned to the normal ranges. Meanwhile, a dry spell had resulted in the pastures changing from lush damp growth with numerous visible fungal colonies growing in the litter to dry firmer material with fewer obvious fungal colonies.

15 November: Orokonui. B631, a 3 year old female wapiti hybrid was reported in very poor condition. In September this was one of 4 animals which developed a scour and lost condition. They were all treated with Ivomec, copper needles and long acting penicillin. The other three recovered. This animal continued to decline. Now in extremely thin condition. Euthanased.

P.M.: Abomasal lining had numerous white nodules up to 3 mm diameter in anterior portion. Posterior portion showed reddening and yellowing of mucosa, with bile in the contents. Other organs relatively normal grossly. Histopathology showed small larvae in the mucosa with foci of mucosal hyperplasia. Mild catarrhal enteritis in large intestine. Mild centrilobular fatty change with shrunken cords towards the portal tracts in the liver. Seronegative for Johnes CFT. Biochemical changes included slightly raised GGT and low serum albumin (see Table 1). Liver copper was 41 μmol/kg (low) and serum pepsinogen was normal (0.44 u/l). Abomasal and intestinal worm counts were negative.

Diagnosis: Parasitic damage to abomasum and lower liver copper levels.

15 November: Orokonui. Four other wapiti hybrid hinds aged 2 to 7 years were in poor condition and had slight scour. Faecal samples showed 1 hind with 1,800 Strongyle e/g, while the other 3 were negative. Serum pepsinogen tests showed a different animal with a level of 1.01 while the other 3 were not elevated. Serum biochemical tests showed low albumin levels in the 2 animals with either elevated egg count or pepsinogen levels. Enzyme levels were in the normal ranges (see Table 1).

Diagnosis: Gastro-intestinal parasites (?). The animals were treated with Ivomec, copper and vitamins and their condition improved.

Discussion

Firstly, it must be emphasised that none of the above cases, nor any of the other cases of fading elk or chronic ill-thrift in elk that have been reviewed recently by Orr et al (1990) should be confused with the condition that occur in elk and mule deer in Colorado and

Wyoming, USA, called "chronic wasting disease". This latter disease, which is typified by progressive weight loss and altered behaviour, is a typical spongiform encephalopathy and probably has the same aetiology as bovine spongiform encephalopathy (BSE) and scrapie which occur in the UK. For this reason it would be wise to avoid terms such as "wasting elk" or "wapiti wasting syndrome". It is preferable to call this the "fading elk syndrome", "wapiti or elk ill-thrift syndrome" or "ill-thrift syndrome in elk".

The main reasons for presenting this case-study were to draw attention to the variety of diseases that may lead to progressive weight loss and to generate discussion about this problem which is widespread in the industry. Our experiences on Invermay deer farms are not unique and are reflected in the wide variety of cases in the lower South Island from which material was submitted to the Invermay Animal Health Laboratory which were reviewed by Orr et al (1990). In the 6 months of this present study the diagnoses included abomasal impaction, sublingual abscess, abomasal parasitism, liver disease, low copper levels and hypoplastic thyroid. In the review of lower South Island cases Orr et al found that thickened abomasal wall and parasitism were the most common necropsy and histopathological findings. Most cases had low serum albumin levels, and low copper levels were found in a high proportion of cases. Other diagnoses included a uterine fibroma in a hind and bronchopneumonia and enteric yersiniosis in 2 weaners. Brain histology in 4 cases revealed no significant findings and serological tests for Johnes disease in 5 cases were negative. Similarly in the present case-study brains and intestines were examined from all necropsied animals and showed no evidence of BSE-like lesions or Johnes disease.

Causative factors

It is curious that elk appear to be more susceptible to chronic-ill-thrift and fading than red deer. There is some evidence that they are more susceptible to copper deficiency, ryegrass staggers and parasites than red deer (Mackintosh, 1990). Whether their susceptibility to parasites is primary or secondary to some other factor which lowers their immunity is not known. They also appear more susceptible to nutritional scour (Bringans, 1987) and this may be related to the pasture types and management systems on New Zealand farms.

There are likely to be a number of contributing factors which may include nutrition (pasture species, stage of growth, palatability, lack of browse, absence of tannin and other plant factors), management (stocking rate, rotational versus set stocking, grazing policies, competition with red deer, time of weaning, stress factors), parasite control (stocking rate, anthelmintic type and frequency of use, grazing management), trace elements (copper, iodine, selenium, cobalt), toxic factors (eg, fungal toxins in pasture including ryegrass staggers, endophyte toxins, sporidesmin and hepatotoxic substances) and infectious agents (Mycobacterium bovis, M. paratuberculosis, M. avium, Fusobacterium necrophorum, etc).

The occurrence of abomasal impaction in cattle is thought to be related to the ingestion of poor quality roughage, especially chopped or ground, and cereal grains. The elk P14, was being fed good quality lucerne hay and crushed barley and the impaction followed a period of diarrhoea which could have reduced rumen function leading to incomplete digestion of the lucerne. The cause of the abomasal torsion and impaction in P29 is not known, but could have been related to feed factors.

Prevention

Because of the complex aetiology it is difficult to make blanket recommendations for prevention of this syndrome. However, much of the following advice is based on our experiences at Invermay and on advice given by Bringans (1987), Pearse (1988) and van Reenen (1989).

Attention should be given to:

a) Improving the nutrition of elk. They should not be treated simply as large red deer. Grazing management and nutrition should be tailor-made for elk. In particular, consideration should be given to finding alternatives to ryegrass, stocking rates should be reduced, longer pastures and browse provided, lucerne hay made available during the spring flush of pasture growth, and elk, hybrids and red deer grazed separately. Weaning of elk calves after the rut may improve their growth rates (J. Haigh, pers. comm.).

b) A parasite control programme. It appears that elk are more susceptible to gastro-intestinal parasites than red deer and therefore they may require anthelmintic treatment more frequently and the faecal egg counts of older animals should be regularly monitored. Elevated serum pepsinogen levels and eosinophilia may also indicate Strongyle larval damage to the abomasum. Utilisation of hay aftermath, reduced stocking densities and grazing red deer to clean up after elk may also reduce parasite problems.

c) Trace element supplementation. The trace element status of pasture and deer should be determined for each property and appropriate supplementation given, bearing in mind that the copper requirement is greater for elk than red deer. Iodine, and perhaps selenium and cobalt should also be considered.

d) Minimising fungal toxic factors. If perennial ryegrass is used then it should be predominantly low endophyte. Northern areas should control sporidesmin build-up in pastures. Experience at Invermay suggests that grazing lush spring pastures can be associated with hepato-toxic damage and scouring. The cause is currently under investigation. Provision of lucerne hay at this time can alleviate the problem.

e) Eliminate infectious agents. Bovine tuberculosis and Johnes disease are the most obvious agents which must be eliminated. Liver or lung abscesses may be difficult to diagnose but they are usually secondary to foot abscesses. Prevent injuries by care with yard and race design.

Treatment

Outbreaks or individual cases of fading elk should be investigated to determine the aetiology if possible. This involves taking a history (nutrition, management, parasite control, trace element supplementation, history of injury, infection, mating, Tb status, etc), careful physical examination and the taking of scour or faecal samples (egg count.

lungworm counts, bacteriological culture, smear for AFO's etc) and blood samples (pepsinogen, serum copper, sick ruminant screen, fibrinogen, haematology).

Severe gastro-intestinal parasitism - Ivomec is the anthelmintic of choice and experience has shown that it may require higher than normal dose rates (2 or 3 times). N.B. Do not use with Se added. The anthelmintic may have to be repeated in 3 to 4 weeks. White drenches have failed to clear abomasal parasites in my experience (unpub. C. Mackintosh).

Copper deficiency - Supplementation can be provided by copper oxide needles or copper injection (painful and may cause a skin slough).

Infection - Treat with appropriate antibiotics.

Nutritional scour - Electrolytes, gut protectants, parenteral antibiotics, B-vitamins, probiotics such as All-Lac and Yeasac all help to restore rumen function.

Severe weight loss - After treating the initial cause the animals have often lost considerable weight and seem to continue down a progressive slide to emaciation. A number of treatments have been used with varying success. We routinely now use: B-vitamins, anabolic steroid (eg, Finajet), probiotics (All-Lac and/or Yeasac), trace element mixes, iodine (Lipiodol) and we provide a variety of feed which may include crushed barley, high protein pelleted concentrate, lucerne hay, red clover hay and cut browse (willow, poplar, pine needles, etc).

Nevertheless trying to treat fading elk can be a humbling and frustrating experience.

References

Bringans, M. (1987): Wapiti health. Deer Farmer, November 1987: 43-44.

Mackintosh, C.G. (1990): Observations on the relative susceptibility to disease of different species of deer farmed in New Zealand. Proceedings Biology of Deer, Mississippi (in press).

Orr, M.B.; Mackintosh, C.G.; Walker, G. (1990): Chronic ill-thrift in elk. Surveillance 17(1): 11-12.

Pearse, A.J. (1988): Wapiti and hybrids - special management needs. Deer Branch NZVA Course No. 5: 164-177.

van Reenen, G. (1989): The "wapiti wasting" syndrome. Deer Farmer, October 1989: 25-26.

Table 1: **Biochemical test results from blood samples taken from clinical cases of "fading" elk and elk-red hybrids at Waiora and Orokonui farms**

	GLDH	SGOT	GGT	TP	Alb.	Haptoglobin (mg/ml HbBC)	Bilirubin
"Normal" ranges	<10(?) u/l	29-132 u/l	14-21 u/l	55-71 g/l	34-50 g/l	<0.2 normal 0.2-0.3 suspicious >0.3 acute inflammation	<12 µmol/l
WAIORA							
BY 724 (2/10) (jaundice euthanased)	15	ND	177	40	16	ND	93
BY 727 (6/10) (poor condition)	43	ND	41	62	28	ND	11
BY 730 (6/10) (poor condition)	2	ND	24	69	27	ND	4
BY 728 (6/10) (poor condition)	9	ND	17	51	25	ND	6
BY 647 (6/10) (poor condition)	3	ND	17	63	28	ND	3
BY 733 (6/10) (sick)	8	ND	181	42	16	ND	17
(euth 21/12)	2	62	22.9	47	15	ND	ND
P 806 (died 16/10)	52	616	237	41	12	2.1	ND
P 802							
(sick 17/10)	87	248	108	46	23	0.99	ND
(conval 25/10)	14	196	143	47	24	0.73	ND
(conval 31/10)	6	ND	102	54	26	1.23	7
(conval 14/11)	20	102	77	51	27	0.56	2
OROKONUI							
B 631 (euth 15/11)	2	ND	31	55	26	ND	11
B 731 thin/scour (15/11)	5	ND	16	44	17	0.86	10
B 526 thin/scour (15/11)	5	ND	17	68	35	0.13	2
B 202 thin/scour (15/11)	5	ND	28	66	37	0.17	3
B 432 thin/scour (15/11)	9	ND	52	65	28	0.47	4

ND = Not Done

Table 2: Biochemical test results from blood samples taken on 19/10/89 and 10/11/89 from elk, elk-hybrid, Pere David hybrid and red deer herd mates of two fading elk P802 and P806

	GLDH	SGOT	GGT	Creat.	TP	Alb.	Haptoglobin (mg/ml HbBC)	
"Normal" ranges	<10(?)u/l	29-132 u/l	14-21 u/l	72-122 u/l	55-71 g/l	34-50 g/l	<0.2 normal 0.2-0.3 suspicious >0.3 acute inflammation	
Pure wapiti								
P 801	12*(2)	82 (55)	38 (19)	101 (102)	56 (60)	36 (40)	<0.2	(0.16)
804	10 (2)	84 (47)	29 (22)	111 (111)	60 (64)	38 (41)	<0.2	(0.15)
805	3 (1)	63 (60)	29 (20)	101 (105)	56 (60)	37 (38)	<0.2	(0.65)
Seven-eighths								
Y 628	3 (2)	56 (51)	22 (19)	97 (95)	60 (61)	35 (38)	<0.2	(0.19)
630	3 (2)	67 (53)	24 (22)	97 (97)	61 (67)	34 (38)	0.2	(0.18)
Three-quarter								
BY 827	2 (2)	52 (52)	19 (18)	111 (109)	56 (59)	33 (36)	0.21	(0.18)
816	6 (2)	93 (85)	24 (25)	99 (111)	60 (63)	35 (38)	0.51	(0.21)
814	3 (2)	49 (52)	22 (19)	140 (121)	60 (61)	36 (38)	0.2	(0.18)
Half								
BY 801	4 (1)	65 (57)	24 (24)	99 (102)	60 (67)	36 (38)	0.22	(0.21)
804	4 (2)	71 (72)	36 (25)	94 (97)	61 (67)	37 (41)	<0.2	(0.16)
805	1 (2)	61 (67)	19 (19)	82 (92)	57 (63)	33 (37)	0.21	(0.19)
Quarter								
BY 838	4 (ND)	52 (ND)	24 (ND)	86 (ND)	62 (ND)	33 (ND)	<0.2	(ND)
834	2 (2)	47 (46)	19 (20)	99 (102)	58 (64)	31 (34)	<0.2	(0.19)
831	5 (2)	58 (62)	40 (33)	86 (93)	66 (70)	35 (36)	<0.2	(0.21)
PD/Red								
WR 899	2 (1)	31 (38)	20 (23)	88 (99)	56 (64)	33 (36)	ND	(0.37)
Red								
G 756	2	38	25	96	70	30	1.31	
755	2	47	27	94	62	31	<0.2	
752	3 (ND)	35 (ND)	19 (ND)	100 (ND)	57 (ND)	29 (ND)	<0.2	(ND)
718	1	31	17	111	59	30	<0.2	
714	2	38	18	100	60	34	<0.2	

ND = Not Done